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Changes in Antagonist Muscles' Coactivation in Response to Strength Training in Older Women

Maarten D. de Boer,[1,2] Christopher I. Morse,[1] Jeanette M. Thom,[1]
Arnold de Haan,[1,2] and Marco V. Narici[1]

[1]Instititute for Biophysical and Clinical Research into Human Movement (IRM),
Manchester Metropolitan University, Alsager Campus, United Kingdom.
[2]Institute for Fundamental and Clinical Human Movement Sciences (IFKB), Vrije Universiteit,
Amsterdam, The Netherlands.

Background. The purpose of this study was to assess changes in neuromuscular function of the plantarflexor and dorsiflexor muscles after 1 year of strength training of these muscles in elderly women. Twelve participants were assigned to a training (74.2 ± 3.1 years) group and eight to a nontraining group (73.6 ± 4.3 years).

Methods. Isometric maximum voluntary contractions (MVC) and muscle activation based on surface electromyography (EMG) were recorded before and after the 12-month training program at six different joint angles.

Results. After training (in the training group), (a) plantarflexion (PF) MVC increased on average by 14.4% ($p < .05$) across ankle joint angles from –20° dorsiflexion (DF) to +30° PF, (b) DF MVC decreased by 5.7% ($p < .05$), (c) PF EMG root mean square increased on average by 22.3% ($p < .05$), and (d) PF antagonists' coactivation increased on average by 7.5% across the tested joint angles. No changes were observed in the nontraining group.

Conclusions. The present results show a significant increase in antagonist muscle coactivation with strength training in older women. The hypothesis is put forward that with a training-induced gain in agonist muscles' torque, stabilization of the ankle joint by increasing antagonist coactivation is needed because of a changed ratio of maximal PF torque to maximal DF torque.

SARCOPENIA, the age-related loss of muscle mass, and muscle weakness are main factors contributing to the loss of functional mobility, independence, and increased frailty in old age (1,2). Maximum muscle strength peaks between the second and the third decade and starts to decrease thereafter, particularly after the sixth decade (3–6). This phenomenon is particularly problematic in women, who, because of starting with a lower muscle mass in young adulthood, are more likely to approach the minimal levels of muscle size and strength necessary for daily activities earlier than are men (7). Besides, decreased strength may lead to an increased risk of falls (8,9), hip fractures (10,11), and loss of bone mineral density (12). Women should thus be the first target group in intervention and rehabilitation studies.

The age-related decrease in strength and power may also be the result of changes in neural drive. Decreased motor unit (MU) recruitment of agonist muscles and decreased MU firing rates (13–16) have indeed been reported in older individuals. In addition, antagonist muscle coactivation appears to be increased in the elderly population (16,17). As suggested by Hortobágyi and DeVita (18), complex interactions between cortical, spinal, and possibly subcortical mechanisms contribute to the changes in coactivation with aging. Increased coactivation contributes to a decrease in net joint torque, but may also protect and stabilize the joint during forceful contractions (19). From a clinical point of view, high antagonist coactivation may be advantageous, as an even pressure distribution across the contact surfaces of the joint seems important in reducing joint degeneration (19,20).

In contrast, strength training in older people can slow down or reverse the decline of muscle mass and function and lead to substantial strength gains. The generally accepted view is that training-induced increases in muscle strength are associated with a reduction in coactivation, increasing net joint torque and reducing energy expenditure (16,17,21,22). However, results from a series of studies showing an increase in elbow extensor coactivation after elbow flexion training (23,24), or no change in coactivation at all (25,26), seem to conflict with this school of thought. Thus, the effect of training on coactivation seems to be an open issue, as also suggested by Gabriel and colleagues (27). Furthermore, little information exists on the neuromuscular adaptations of older people to training periods lasting longer than a few months (28), and only a limited number of studies looked at the effects at multiple joint angles (29), despite the notion that strength gains are angle-specific (30,31).

In light of these considerations, a longitudinal study was organized to assess the neuromuscular adaptations of older women to a 1-year training program. In the present study, we report the changes in maximum torque and neural drive of the plantarflexor and dorsiflexor muscles at various ankle angles. Based on previous literature, we hypothesized that 1 year of progressive strength training would result in (a) an increase in maximal isometric strength of the plantarflexor muscles, (b) an increase in activation based on electromyography (EMG) root mean square (RMS), and/or (c) a decrease in antagonist muscles' coactivation in elderly females.

Methods

Participants

Twenty-six healthy community-dwelling, independent older women participated in this study. Fourteen were assigned to a training group (age 74.2 ± 3.1 years, height 1.6 ± 0.6 m, weight 69.7 ± 18.6 kg, mean ± standard deviation [*SD*]), and twelve to a nontraining control group (age 73.6 ± 4.3 years, height 1.6 ± 0.4 m, weight 61.4 ± 9.4 kg). All participants were medically screened to exclude cardiovascular, myopathic, neurological, neoplastic, and/or inflammatory diseases. All were physically active, but not engaged in competitive sports. All procedures were approved by the Ethics Committee of the Manchester Metropolitan University, and informed consent was obtained from each participant. The participants were familiarized with all proceedings on a separate session before data collection.

Training Protocol

The 12-month training protocol consisted of combined aerobic, weight-lifting exercises mainly of the knee extensors and plantarflexor muscles, stretching, and Tai Chi exercises performed three times per week. Two sessions were supervised in a gym and one was performed at home. Training of the lower limb muscles involved bilateral knee extension performed on a leg-press and on knee-extension machines, and, for the PF, on a sitting calf-rise machine (Technogym, Gambettola, Italy). During the first 6 months, training consisted of two series of 8–10 repetitions at 80%–100% of the eight repetition maximum (8RM). After the initial 6 months, the volume of the training was increased to three series of 8–10 repetitions at the 8RM. The training load was reviewed and updated at the start of each month to match it to 80%–100% of the 8RM. Each training session was preceded by an aerobic warm-up exercise, and followed by stretching and Tai Chi exercises.

Strength Measurements

Isometric plantarflexion (PF) and dorsiflexion (DF) were recorded with the participants prone, and with the left foot secured to the adapter of an isokinetic dynamometer (Cybex Norm; Cybex International, Ronkonkoma, NY). The participants were positioned with the knee fully extended and the lateral malleolus aligned with the axis of rotation of the dynamometer. The foot was tightly secured to the footplate to minimize heel displacement, and the participants performed three submaximal isokinetic PF–DF contractions as a warm-up. Maximal voluntary isometric contractions (MVC) were tested at six joint angles: 20° and 10° DF, and 0°, 10°, 20°, and 30° PF. Two PF MVCs were performed at each angle in randomized order, and then a DF MVC was performed at each angle, again randomized. Trials were interspersed by a 2-minute rest. The same procedures were applied when testing after 1 year of training.

EMG Measurements

During each MVC, surface EMG activity of the gastrocnemius medialis (GM), gastrocnemius lateralis (GL), and tibialis anterior (TA) muscles was recorded using two pregelled 10-mm Ag-AgCl unipolar electrodes (interelectrode distance 25 mm; Medicotest, Ølstykke, Denmark), after skin cleansing with abrasive gel to reduce interelectrode impedance below 5 kΩ (XI-1 Electrode impedance tester; Oxford Medical Ltd, Elk Grove Village, IL). Muscle boundaries were identified using ultrasonography to reduce the influence of cross-talk, and electrodes were placed along the midsagittal axis of the muscle belly at the proximal third part between the motor point and myotendinous junction. Reference electrodes were placed over the lateral epicondyle of the femur. Anatomical landmarks were used to reproduce the same electrode location in successive testing sessions. A raw EMG was acquired with a sampling frequency of 2000 Hz, and processed with a multi-channel analogue-digital converter (EMG 100B; Biopac Systems, Santa Barbara, CA). The raw signal was filtered using analogue high-pass (10 Hz) and low-pass (500 Hz) filters, and was amplified with a gain of 2000. Coactivation of the GM, GL, and TA was assessed using the RMS of the EMG signal, calculated over 1.0 second around the peak MVC torque. The ratio of RMS during antagonist MVC to RMS during agonist MVC was multiplied by 100 and was used as a measure of coactivation (16).

Statistics

To determine the effect of the training program and the different joint angles on the PF and DF MVC torque, maximal voluntary activation, and coactivation of the GM, GL, and TA muscles, a repeated-measures analysis of variance (ANOVA) was conducted with $\alpha = 0.05$. Bonferroni post hoc adjustment was used for further analysis. The dependent variables are PF and DF peak torque, plantarflexor and dorsiflexor EMG RMS, and coactivation of the GM, TA, and GL muscles. The independent variables are joint angle (–20°, –10°, 0°, 10°, 20°, and 30°), the conditions pre- and posttraining, and muscle (GM, GL, and TA). Unless stated otherwise, results are means ± *SD*.

Results

Torque

Plantarflexion.—Training resulted in a significant increase in PF MVC torque at 20° and 10° DF ($p < .01$ and $p < .05$, respectively) and at 10° PF ($p < .05$), with a mean increase over angles of 11.7% ($p < .01$, Figure 1A). At the optimum angle of 20° DF, PF torque increased by 18.4% (from 82.7 ± 26.3 to 97.8 ± 36.0 Nm, $p < .01$).

No significant differences in PF MVC torque (Figure 1A) were found in the control group. No significant difference between body masses in the two groups existed ($p = .3$). As expected, normalizing torque to body mass did not change the main effects found without normalization.

Dorsiflexion.—Training resulted in a significant decrease in DF MVC torque at 20° and 30° PF ($p < .01$ and $p < .001$, respectively, Figure 1B) with a mean decrease over angles of 5.7% ($p < .05$). No significant changes were found in the control group.

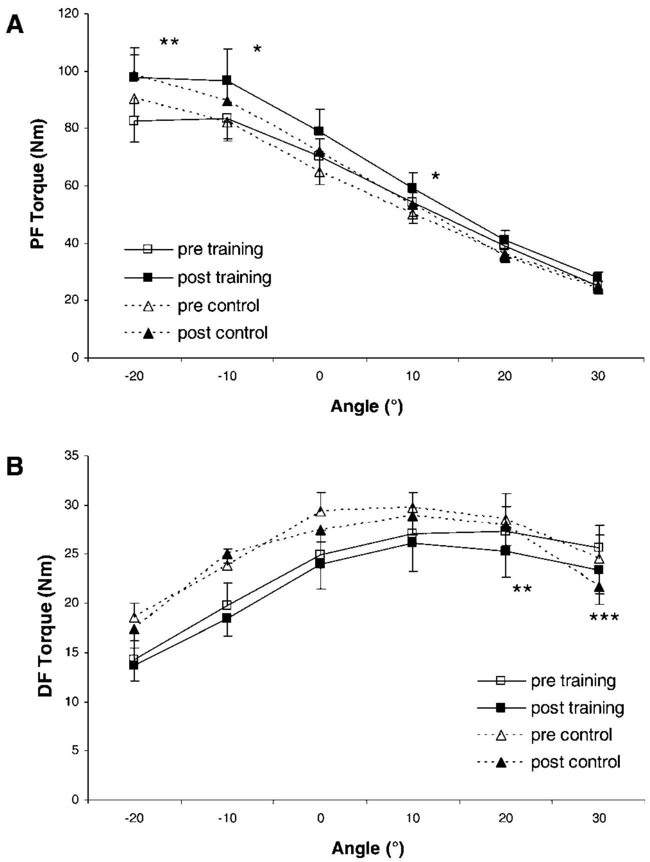

Figure 1. Pre- and posttraining plantarflexion maximum voluntary contraction (PF MVC) (**A**) and dorsiflexion (DF) MVC torque (**B**) versus joint angle. Values are means ± standard error. *Significantly different between pre- and posttraining, $p < .05$; **$p < .01$; ***$p < .001$. Lines are added for clarity.

EMG

Although a tendency for an increase in EMG activity (RMS) of the GM muscle of 10.3% (Figure 2A) and a decrease in that of the TA and GL muscles of 11.6% (Figure 2B) and 2.1%, respectively (Figure 2C), were observed during DF, these changes were not significant. Training resulted in a significantly higher RMS value for the GM muscle during PF (mean increase 21.3%, $p < .05$, Figure 3A), and also for the TA and GL muscles (42.6%, $p < .005$, Figure 3B and 22.9%, $p < .001$, Figure 3C, respectively). At the optimum angle for PF torque, the GM, TA, and GL RMS, respectively, increased by 18.4%, 47.2%, and 26.2%. The controls showed no significant changes.

Coactivation

After training, TA coactivation significantly increased at 20° and 10° DF ($p < .05$) and at 0°, 10°, and 20° PF ($p < .05$, Figure 4A), with a mean increase of 7.5%. At the optimum PF angle, the increase in DF coactivation was 7.4% (from 11.9 ± 7.6% to 19.3 ± 11.0%).

No differences between pre- and postconditions were found in the GM coactivation during DF MVC (mean decrease was 1.7%, not significant, Figure 4B). However, training resulted in a decreased GL coactivation during DF

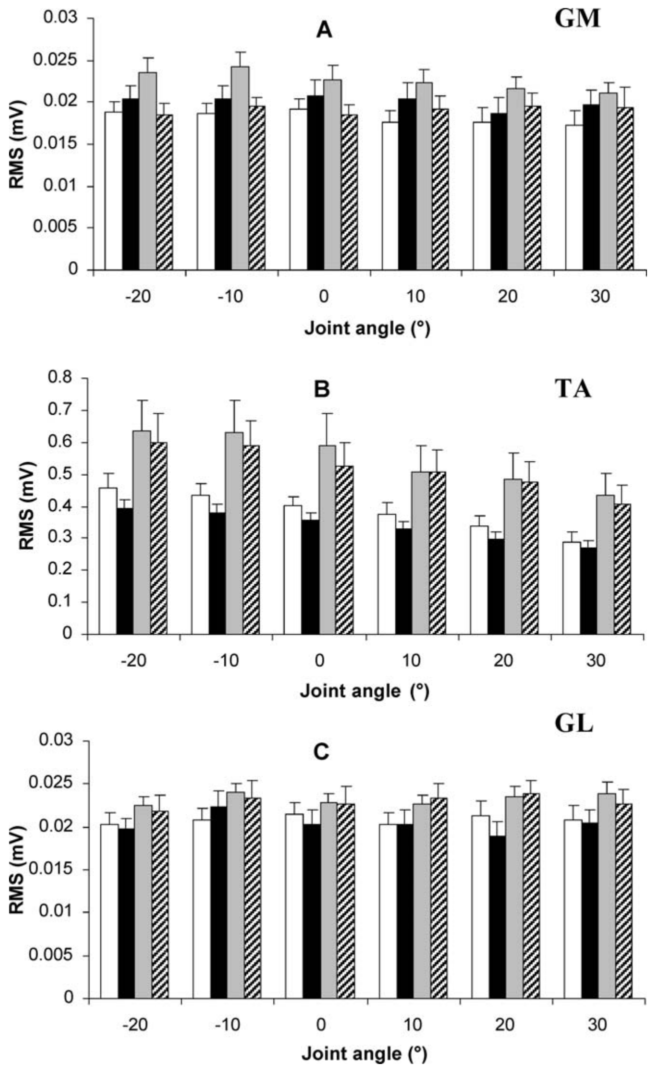

Figure 2. Root mean square (RMS) values during dorsiflexion maximum voluntary contraction (DF MVC) for gastrocnemius medialis (GM) (**A**), tibialis anterior (TA) (**B**), and gastrocnemius lateralis (GL) (**C**) muscles, at six different joint angles. Values are means ± standard error. *White bars:* pretraining; *black bars:* posttraining; *gray bars:* precontrol; *hatched bars:* postcontrol.

MVC at 10° DF, and at 0° and 20° PF ($p < .05$, Figure 4C), with a mean decrease of 4.0% ($p < .01$). No significant changes were found in the control group.

Discussion

The present study showed that a 1-year training program in older women resulted in: (a) a marked increase in PF MVC torque, (b) a decrease in DF MVC torque, (c) an increase in TA coactivation, and (d) a decreased GL coactivation after training. Moreover, there was an angle effect both for PF and DF MVC and RMS, and for GL coactivation during DF MVC.

The maximal strength gains during PF were accompanied by significant increases in PF RMS. Surface EMG is more likely to pick up action potentials from larger MUs, consisting mainly of type II fibers (32,33), located more

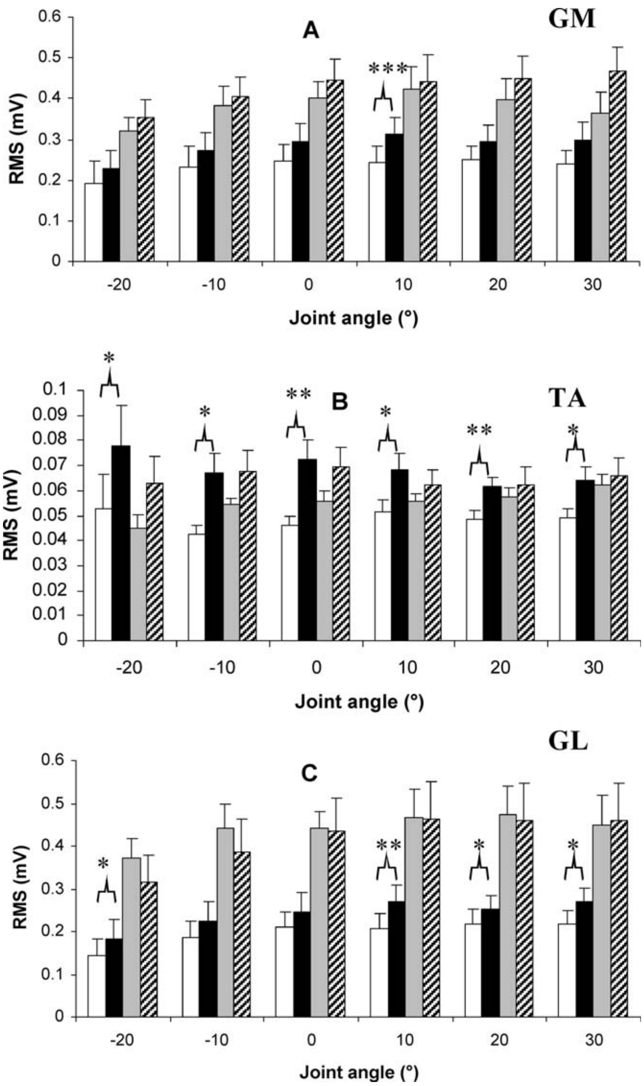

Figure 3. Root mean square (RMS) values during plantarflexion maximum voluntary contraction (PF MVC) for gastrocnemius medialis (GM) (**A**), tibialis anterior (TA) (**B**), and gastrocnemius lateralis (GL) (**C**) muscles, at six different joint angles. Values are means ± standard error. *Significantly different between pre- and posttraining, $p < .05$; **$p < .01$; ***$p < .005$. *White bars:* pretraining; *black bars:* posttraining; *gray bars:* precontrol; *hatched bars:* postcontrol.

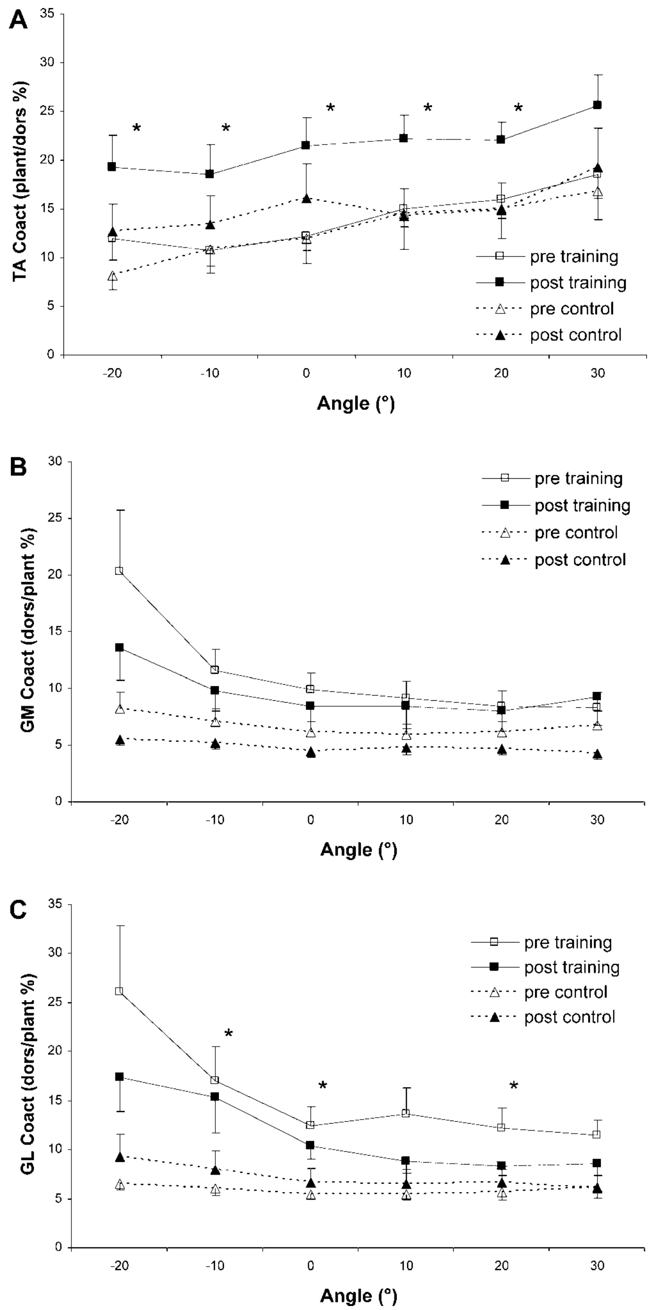

Figure 4. Coactivation of the tibialis anterior (TA) (**A**), gastrocnemius medialis (GM) (**B**), and gastrocnemius lateralis (GL) (**C**) muscles versus joint angle. Coactivation was calculated as the ratio of root mean square (RMS) during antagonist maximum voluntary contraction (MVC)/RMS during agonist MVC multiplied by 100 (%). Values are means ± standard error. *Significantly different between pre- and posttraining, $p < .05$. Lines are added for clarity. Coact = coactivation; dors = dorsiflexor; plant = plantarflexor.

superficially in the muscle. Knight and Kamen (33) found an inverse relationship between recording depth and maximum peak-to-peak amplitude, indicating larger action potentials for MUs with mainly type II fibers. Taken together, these data suggest that, besides increased MU synchronization and higher firing frequencies, the present increase in EMG RMS might be partially due to hypertrophy of type II fibers. The 12% increase in PF MVC torque after training is in agreement with a 12.4% mean increase in PF MVC found by Ferri and colleagues (29). In line with the results of Scaglioni and colleagues (34), PF MVC at 20° DF increased by 18.4% ($p < .01$). However, if RMS EMG values are compared to the largest increase in PF torque, occurring at the optimum angle, we see that these values are lower at this angle both pre- and posttraining, suggesting that other mechanisms may account for the increases in maximal isometric strength. The obvious candidate is muscle hypertrophy. Although PF muscle size is not yet available for these elderly women, in aged-matched men who underwent the same training protocol, PF muscle volume increased by 12.2% (25). It seems likely that the present group of older women would display a similar hypertrophy, and the size of this increase well matches that of PF MVC (12%). The decrease in DF MVC torque is more difficult to explain. The training protocol mainly aimed at the plantarflexors, but no decrease in DF MVC torque was found in the control group. Several factors might have contributed to this decrease. A decrease in TA activation, as indicated by the 11.6% decrease in TA RMS is a possible candidate; however, this decrease was not significant. A decrease in TA cross-sectional area CSA might have occurred; however, this is very unlikely after training. Possibly, the decrease in DF torque could be the effect of a relatively higher coactivation of the plantarflexors, despite the observed decrease in GM coactivation after training. A lower absolute coactivation of the plantarflexor muscles might be needed because of the decrease in DF MVC torque, but the decrease in GL coactivation could be relatively small, leading to a relatively higher GL coactivation than before training. This seems to be the case indeed as the relative decrease in DF torque was almost three times greater than the relative decrease in GL coactivation after training (mean decrease over 20° and 30° PF of 9.3% in torque vs 3.4% in coactivation). Also, the different training protocol for the DF and PF muscles could have led to an imbalance in the contribution of central and peripheral neural pathways that control torque generation.

TA activation in terms of RMS EMG values during PF MVC increased significantly, implying an increased coactivation, as TA RMS during DF MVC remained unaltered. This increase in coactivation may be a response to the increase in maximal PF MVC torque. It has been suggested that coactivation is facilitated by Renshaw's cell firing, which inhibits Ia-inhibitory interneurons (35) by excitation of Ib interneurons from the Golgi tendon organs (20,36,37) or by direct descending motor pathways (20,37), and is indeed controlled by a complex interaction between central and peripheral mechanisms (18). In this study, increased coactivation could be seen as a consequence rather than a cause of increased net joint torque. Stabilization of the ankle joint through increased coactivation might be necessary because of a changed ratio of maximal PF to DF torques. Considering coactivation as an adaptation to a changed torque ratio over the ankle joint might also explain the decrease in GL coactivation during DF MVC. However, closer investigation of this hypothesis did not reveal an increased coactivation with an increase of maximal antagonist torque to maximal agonist torque ratio. In contrast, the increased coactivation of the TA during PF MVC was uniform across all of the tested joint angles, implying a central component in the control of coactivation.

Conclusion

The present results show a significant gain in plantarflexor muscle torque, accompanied by an increase in antagonist muscle coactivation, induced by strength training in old age. The increase in PF MVC torque was associated with increased activation based on EMG RMS data. Furthermore, the decrease in dorsiflexor torque was associated with a decrease in PF coactivation. The hypothesis is put forward that stabilization of the ankle joint is needed in the elderly population because of a changed ratio of maximal PF torque versus maximal DF torque.

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Correspondence

Address correspondence to Professor Marco Narici, PhD, Institute for Biophysical and Clinical Research into Human Movement (IRM), Manchester Metropolitan University, Alsager, ST7 2HL, United Kingdom. E-mail: m.narici@mmu.ac.uk

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